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Temperature and cardiovascular mortality

Excess deaths from heart disease and stroke in northern Europe are due in part to the cold

Cardiovascular disease is the most common cause of death in most developed countries, but mortality is particularly high in some parts of Britain. These high rates can be lowered only if the causes are understood. Preventive campaigns have concentrated on smoking, hypertension, and hyperlipidaemia. These risk factors explain at most half of all myocardial infarctions.[1]

Deaths and hospital admissions for coronary heart disease and stroke are higher in winter than in summer in many temperate countries.[2] In some winters mortality has been as much as 70% higher than in the summers. The size of the winter excess is related to the difference in environmental temperature. Excess winter cardiovascular mortality has fallen in recent years, but it remains numerically far more important than other causes of winter deaths such as respiratory infections or hypothermia.[3] Seasonal fluctuations in cardiovascular events are greater in Britain than in some other countries with wider temperature variations and colder winters, and this suggests that the excess winter deaths may be preventable.[2] Lesser seasonal variations may be related to more constant indoor temperatures in those countries. In Britain excess winter mortality is greatest in socially deprived people who have the worst domestic heating and the highest overall mortality.

Seasonal variations in vascular events do not necessarily result in an increase in annual mortality. Deaths in winter might have been going to occur later that year. Excess winter deaths will have a real effect on mortality only if the deaths occur so prematurely that they appreciably shorten life expectancy in people who would otherwise have died from another disease.

In Europe annual mortality from cardiovascular disease increases progressively from Mediterranean countries to Scandinavia. Inherited susceptibility, diet, and prevalence of smoking are obvious predisposing factors which might vary between countries. Big variations in cardiovascular mortality also, however, occur within national boundaries of many European countries and cannot be explained by these factors. Higher rates within countries correlate with increasing latitude and decreasing temperature.[4] Environmental temperature, rainfall, and socioeconomic variations accounted for 41% of the inter-town variance in deaths from coronary heart disease in a study in England and Wales in the 1970s.[5] The standardised mortality ratios for two towns differed on average by 7% for each 1°C temperature difference, while the other two variables had less effect on mortality. The seasonal and geographical data suggest that low environmental temperature has an important effect on cardiovascular mortality.

There are several possible mechanisms. Environmental temperature has an inverse relation with blood pressure.[6] The mean blood pressures of populations in cold parts of the country may possibly be higher than in warmer areas. Certainly the difference between summer and winter temperatures in Britain results in a difference of about 5 mm Hg. Sustained differences in blood pressure of this order are associated with at least a 21% difference in coronary events and at least a 34% difference in stroke.[7] A pharmacological reduction of blood pressure of this amount is associated with a reduction in coronary events by 14% in patients with hypertension and a greater than 20% difference in the rate of fatal myocardial infarction in patients with left ventricular dysfunction.[8,9]

A raised blood pressure in a cold environment has several adverse effects. It alters the ratio of myocardial oxygen supply to demand. It increases the ventricular wall stress, cardiac work, and oxygen requirements, but it reduces mechanical efficiency and may impair coronary blood flow, particularly in people with fixed stenoses. Consequently cold can precipitate myocardial ischaemia.

Peripheral vasoconstriction induced by the cold may cause acute pulmonary oedema by overloading the left ventricle even in people without coronary stenoses, but particularly in those predisposed to hypertension.[10] Since vasodilating drugs have a beneficial effect on survival in patients with impaired left ventricular function, the long term effects of the high preload and afterload induced by a cold environment would be expected to have an adverse effect on survival.

The effects of vasoconstriction in the cold are not confined to the myocardium. The external work done by the heart is dissipated by friction of blood flow through vessels, by shock losses of energy at bends and branch points, and by damping of pulsatile flow by the vessel walls. Unless the heart fails, during peripheral vasoconstriction the main haemodynamic change is increased arterial pressure. Systolic pressure increases more than diastolic so that pulse amplitude is generally increased, but there is usually little change in the cardiac output or the pulse rate.[11] These rises in the blood pressure and pulse pressure increase the forces acting to produce deformation of the vessel wall. The unaltered cardiac output passing through constricted vessels results in greater rates of flow in the small vessels. Shock loss of energy and friction are increased. Shear at the vessel surface increases. The resulting vascular damage may have acute and chronic effects. Arterial dissection may be the initiating event in a number of acute cardiovascular syndromes, including myocardial infarction, unstable angina, some cerebrovascular syndromes, and rupture of the aorta. Hypertension is itself atherogenic. Possible initiating mechanisms include repeated minor injuries to the vessel wall and release of vasoactive substances as a result of increased shear.[12]

The excess number of coronary events in cold climates may also be related to blood clotting. The plasma concentration of fibrinogen is inversely related to the environmental temperature, but part of the rise in fibrinogen concentrations in the winter may be the result of seasonal respiratory infections.[13,14] The seasonal variations in fibrinogen concentrations due to temperature changes may be half that resulting from smoking.[13] In cold conditions the plasma concentrations of some clotting factors are increased, as are platelet count and in vitro platelet aggregation.[11,13-15] A reduced plasma volume and increased blood viscosity during cold exposure also tend to promote thrombosis.[11,15] Whether these factors have a role in atherogenesis is uncertain. Experiments have produced contradictory results on how environmental temperature affects lipid metabolism, but cold can adversely alter plasma lipid concentrations, making abnormal thrombosis more likely and having a potential chronic atherogenic effect.[14]

All these data suggest that at least part of the excess mortality from cardiovascular diseases in some parts of Britain is due to those areas being relatively cold. For the north to achieve reductions in cardiovascular mortality to the rates present in the warmer south will require greater improvements in other risk factors to compensate for the influence of climate. How far social measures such as improvements in poor heating and home insulation would have an impact on cardiovascular mortality in cold parts of the country is uncertain.

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Health implications of putting value added tax on fuel

Time to combat fuel poverty

The American senator Hubert Humphrey said, "The moral test of government is how that government treats those who are in the dawn of life, the children; those who are in the twilight of life, the elderly; and those who are in the shadows of life—the sick, the needy, and the handicapped." By such criteria, putting value added tax (VAT) on fuel is a bad measure.

Almost three quarters of pensioners in Britain pay no income tax, yet virtually all pay VAT on their gas and electricity bills. Only if the government had added VAT to food or water could it have increased the tax burden for an equivalent number of households made up of elderly people.

The tax burden is not evenly distributed.[1] Although households with low incomes spend less on fuel than those with high incomes (spending on fuel being mainly related to the size of the place where you live), the proportion that they spend is substantially higher—13% in the lowest fifth of income compared with 4% in the highest fifth of income. Households made up of single parents with children aged under 5 or single pensioners spend one sixth of their income on fuel. When households in the top and bottom tenths of income distribution are compared the burden of increased fuel costs due to VAT is seven times greater in poor families.

The term "fuel poverty" was coined in the early 1980s to describe the situation in which people who are least able to afford the cost of heating tend to live in houses that are hardest to heat and, as a result, achieve lower indoor temperatures with the fuel that they buy.[1,2] The high prevalence of dampness and condensation shown by national surveys of the condition of housing—one in five households in England and one in three in Scotland are affected—is largely due to the difficulty of maintaining internal temperatures in poorly designed and insulated houses. In the Glasgow housing condition survey half of families with children were living in houses affected by dampness and condensation.[3]

Low income groups also tend to use dearer fuel because of their lack of access to gas central heating, their greater reliance on electricity and bottled gas, and the extra charges made by power companies for prepayment meters and schemes to "ease" payments.[1] All these problems are accentuated by the colder climate and greater cost of maintaining indoor temperatures in the north of Britain.[4]

Pensioners have a greater need for fuel, given the time they spend at home and the decline of body thermoregulation in old age. Ironically, shops can be closed down in Britain if they do not maintain an internal temperature of 16°C, yet 37% of households made up of elderly people